Splenoportagraphy in Portal Hypertension

Its Value in Selecting the Operative Procedure of Choice

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• **Splenoportagraphy has become an almost indispensable adjunct to the surgical management of portal hypertension. In many instances it will provide the basis for the selection of the operative procedure. Certain instances of intrahepatic portal hypertension due to cirrhosis that might better be managed by splenorenal shunt rather than by the generally preferred method of direct end-to-side portacaval shunt may be determined by this procedure. The procedure finds its greatest application in the accurate delineation of the three major types of extrahepatic portal hypertension, each of which demands a different surgical approach.**

X-RAY VISUALIZATION of the portal venous system has become an almost indispensable adjunct to the surgical management of portal hypertension. Splenoportagraphy, when properly carried out, clearly delineates this system and avoids tedious and sometimes hazardous surgical dissection. Moreover, extensive surgical dissection in these cases frequently does not adequately uncover the full extent of the disease process. Splenoportagraphy, in many cases of portal hypertension, will provide the basis for the selection of the operative procedure.

Splenoportagraphy was introduced by Abeatici and Campi in 1951.[1] Their technique, as well as that of others,[3,4] involves the performance of the test in the x-ray department by means of percutaneous splenic injection of radiopaque material under local anesthetic skin infiltration.

It has been difficult for us to understand why more complications have not been reported by this technique. Early in our experience[5] we encountered significant splenic hemorrhage and now employ the procedure only in the operating room after the abdomen has been opened. This permits direct tamponade of the splenic puncture site after completion of the injection. Further, in our experience, a preoperative splenoportagram is rarely required. We do not consider this test to be of significant value in the decision as to whether or not operation is indicated. This information is obtained by the multitude of clinical features that characterize bleeding esophagogastric varices. Thus, its use is primarily for delineating the anatomical features of the portal hypertension and thus determining the operative procedure most likely to succeed. Positioning of the patient and placement of the incision are admittedly facilitated by a preoperative portagram. The lack of this information has, however, not proved a significant handicap.

The recommended initial incision is a transverse upper abdominal one that permits exploration and splenoportagraphy. The incision is then extended into the right or left chest as determined by the

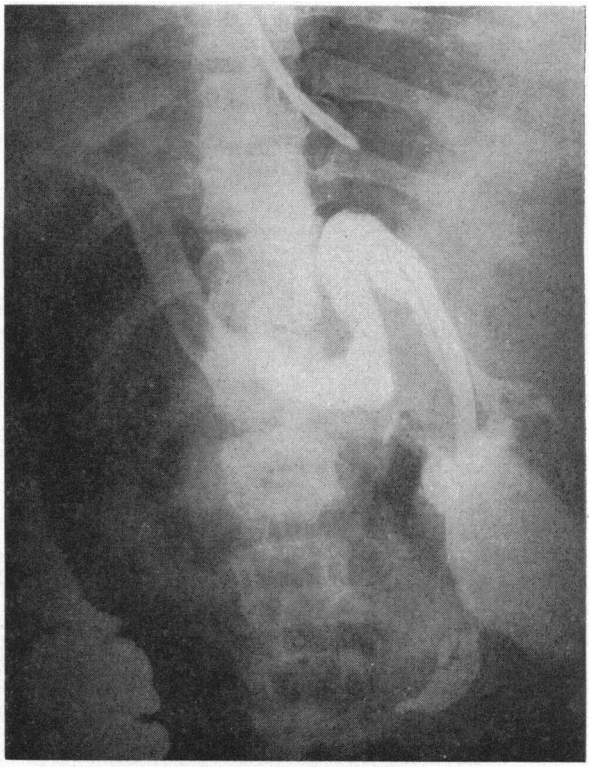

Figure 1.—Splenoportagram of 12-year-old girl with posthepatitic cirrhosis. Massive bleeding from esophagastric varices and pronounced hypersplenism were clinical features. A splenic vein, perhaps unsatisfactory for an adequate splenorenal shunt, is demonstrated.

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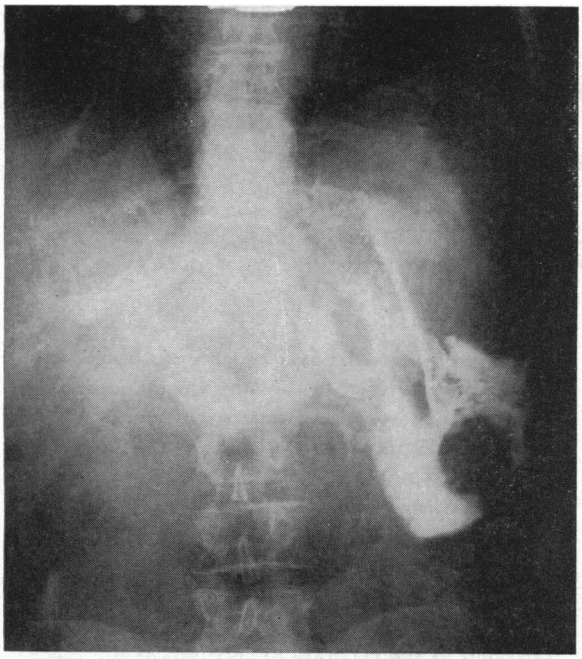

Figure 2.—Splenoportagram of 44-year-old man with posthepatitic cirrhosis. Hypersplenism was the dominant clinical manifestation. Gastrointestinal bleeding was minimal. A splenic vein suitable for splenorenal shunt is demonstrated.

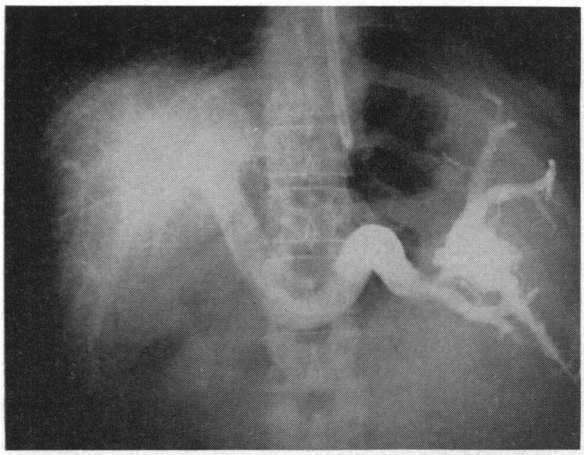

Figure 3.—Splenoportagram of 46-year-old man with posthepatitic cirrhosis. Minimal collateral circulation is noted and the finer portal radicals within the liver are clearly visualized. Complete diversion of the portal blood flow by end-to-side portacaval shunt resulted in a considerable degree of hepatic decompensation.

information obtained. In the course of this presentation, illustrative examples of the need and value of splenoportagraphy will be presented.

INTRAHEPATIC PORTAL HYPERTENSION

Although the surgical procedures designed to alleviate the complications of portal hypertension are not standardized, certain principles of such operations are generally accepted. When the primary etiologic mechanism for the portal hypertension exists within the liver and the entire portal venous tree is patent, an adequate shunt from this venous system to the caval system is the operation most likely to be of benefit. This may be accomplished by a direct portacaval shunt or by the indirect method of splenectomy and splenorenal anastomosis. The relative merits of these two operations will not be argued. Nevertheless, our experience has led us to prefer the direct portacaval shunt which we have now performed in somewhat more than 100 patients. Also we prefer to completely divide the portal vein and accomplish the shunt in an end-to-side manner. We have not employed splenoportagraphy routinely in such cirrhotic patients. Palpation of the hepatoduodenal ligament at the time of operation will usually reveal the portal vein to be taut, distended and patent. If there is any doubt in this regard there is no hesitancy to perform splenoportagraphy during operation.

Occasionally for a patient with intrahepatic portal hypertension who has a patent portal venous tree, a splenorenal anastomosis will be preferred. This situation is encountered when significant hypersplenism as manifested by severe pancytopenia is present and splenectomy would seem desirable. In many such situations, however, it must be recognized that relief of the hypersplenism will occur when portal decompression is accomplished by direct portacaval anastomosis even though the spleen is not removed. The efficiency of a venous shunt in eliminating portal hypertension depends to a great extent upon the size of the afferent vessel and the size of the anastomosis. Thus, in choosing a splenorenal anastomosis over a direct portacaval anastomosis, it is essential that the splenic vein be of appropriate size. Usually the primary purpose of these operations is to alleviate the threat of life-endangering hemorrhage from esophagogastric varices. One must thus weigh carefully these two factors when dealing with a patient with intrahepatic portal hypertension and hypersplenism.

This problem is exemplified by a brief account of two representative patients. In both patients severe pancytopenia secondary to intrahepatic portal hypertension, due to cirrhosis was manifest. The first patient was a 12-year-old girl who had bled massively on two occasions within three weeks preceding operation. Pronounced leukopenia and thrombocytopenia were present. Upon examination of the splenoportagram it was noted that the splenic hilar veins did not fuse to form the common splenic vein for quite a distance from the spleen (Figure 1). This suggested there might be difficulty dissecting out a sufficiently large splenic vein for anastomosis and hence the possibility that a graft might

be required. In our opinion, available autogenous grafts are not of sufficient diameter to guarantee a satisfactory shunt. Our primary concern with this patient was to relieve the portal hypertension and control the bleeding. Thus a direct portacaval shunt was performed and the portal pressure was reduced to a normal range. In five months of observation after operation she had no further bleeding and the depression in circulating cellular elements steadily improved but had not yet risen to normal levels at the time of last observation.

The second patient was a 44-year-old man who also had pronounced pancytopenia and thrombocytopenia. Gastrointestinal bleeding had occurred but had never been massive. A splenoportagram (Figure 2) showed a large splenic vein, eminently suitable for anastomosis. Of major concern in this patient was the control of hypersplenism. A splenectomy followed by splenorenal shunt was decided upon. A rather poor reduction in portal pressure was obtained. The circulating cellular elements returned immediately to normal levels, where they remained. There was no further gastrointestinal bleeding.

Another way in which splenoportagraphic delineation may help in surgical consideration of a case of intrahepatic portal hypertension is in estimating the amount of blood flow through the liver that is contributed by the portal vein. In the usual cirrhotic patient, the total liver blood flow is reduced about 50 per cent. This occurs in spite of the apparent pronounced increase in arterial blood that reaches the liver. The assumption is that reduction in flow of blood through the liver takes place primarily at the expense of the portal flow. It is reasonable to believe that rather significant differences may exist in this regard from one cirrhotic patient to another. If it could be determined beforehand that one was dealing with the unusual cirrhotic patient whose portal vein was contributing the majority of liver blood flow, the surgeon might be reluctant to completely divert this flow from the liver as is done in an end-to-side portacaval shunt.

When a splenoportagram fails to show significant collateral vessels and the finer portal radicals within the liver are clearly visualized, it is a justifiable interpretation that portal flow is rapid and liver obstruction is minimal. It may further be deduced in such a situation that the portal vein is contributing a significant amount of the total flow of blood through the liver. A preoperative indication that this problem may exist is presented when an esophagram or esophagoscopy fails to reveal significant varices in a cirrhotic patient who has bled. We now perform splenoportagraphy routinely in these patients and utilize splenorenal anastomosis if our preoperative suspicions are confirmed. Figure 3 is a splenoportagram in the case of such a patient, in whom a direct end-to-side portacaval shunt was done. Preoperative evaluation indicated this patient to be an excellent surgical candidate. The postoperative course was uneventful for two weeks and the patient was discharged from the hospital. He returned two weeks later with icterus and ascites. Liver function studies demonstrated significant impairment, much greater than might be anticipated from the effects of anesthetic and sur-

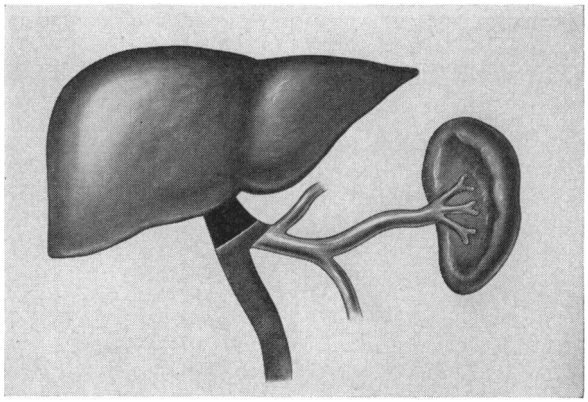

Figure 4.—Extrahepatic portal hypertension: Type No. 1. The portal vein alone or the portal and superior mesenteric veins are occluded. The splenic and coronary veins are patent.

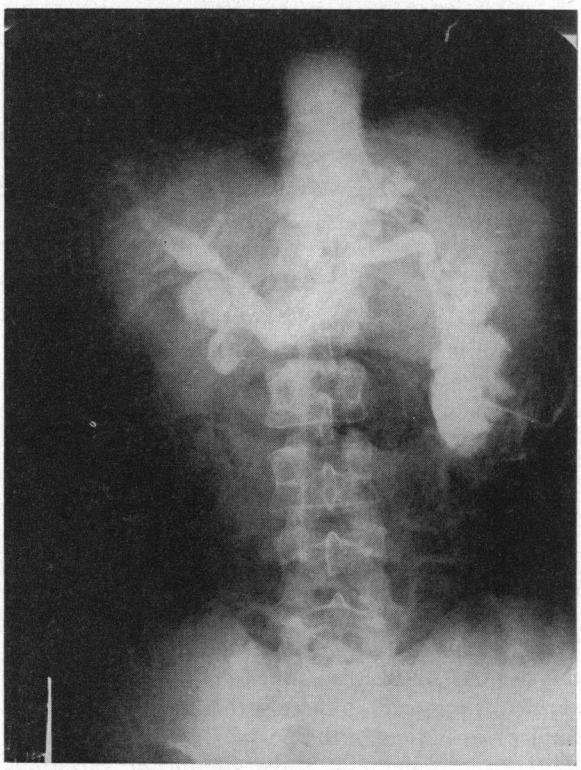

Figure 5.—Splenoportagram of 34-year-old man with decided narrowing of the portal vein due to chronic pancreatitis localized to the head of the gland.

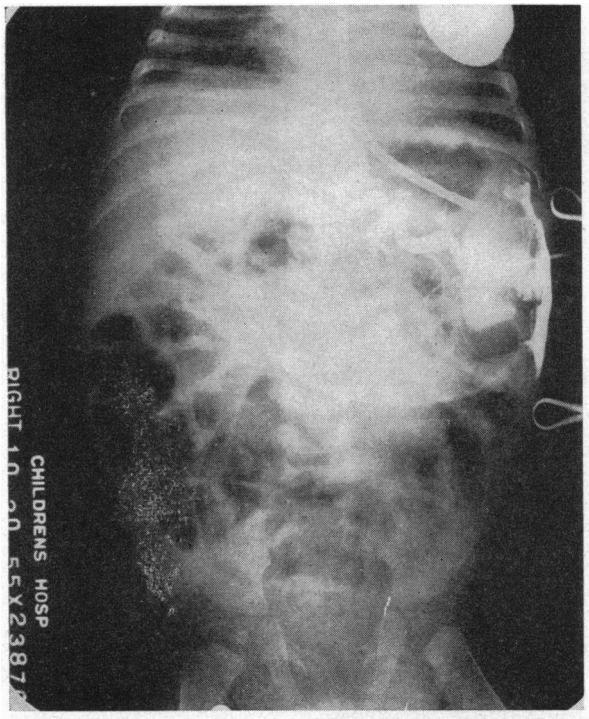

Figure 6.—Splenoportagram of nine-months-old infant. Neither the portal nor the superior mesenteric vein is visualized. A large coronary vein is apparent.

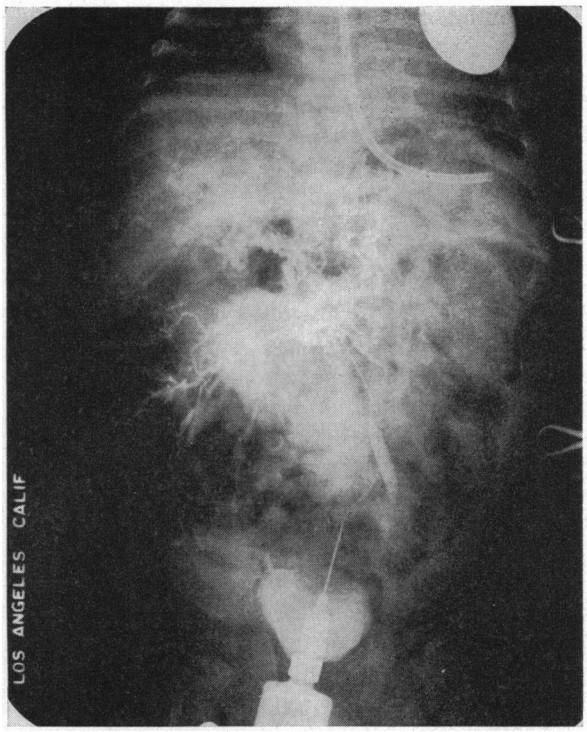

Figure 7.—Jejunal portagram in same infant as shown in Figure 6. Complete obliteration of both the portal and superior mesenteric veins is confirmed.

gical stress alone. The subsequent course was a slow but definite improvement. Perhaps in this patient this degree of hepatic decompensation would not have developed if splenorenal shunt had been done.

EXTRAHEPATIC PORTAL HYPERTENSION

Splenoportagraphy, though valuable in cirrhosis, finds its greatest use in delineation of the disease process in extrahepatic portal hypertension. In this disease, the pathologic process involves obstruction of the portal vein or one or more of its tributaries. The liver itself is normal. The site and extent of this venous obstructive process will determine the surgical approach. Although there are minor anatomical variations in the portal venous tree, from a therapeutic standpoint three significant variations of extrahepatic portal hypertension are encountered. For the sake of convenience, we have elected to label these as types 1, 2, and 3. Minor variations of these types will occasionally be encountered.

Type 1 (Figure 4): The portal vein alone or the portal and superior mesenteric veins are occluded. The splenic and coronary veins are patent. All of the splanchnic return blood flow is shunted to the caval system via inadequate collaterals. In this instance a direct portacaval shunt is impossible. A splenorenal shunt for this problem is the procedure most likely to result in decompression of the esophagogastric varices. Shunts using the superior

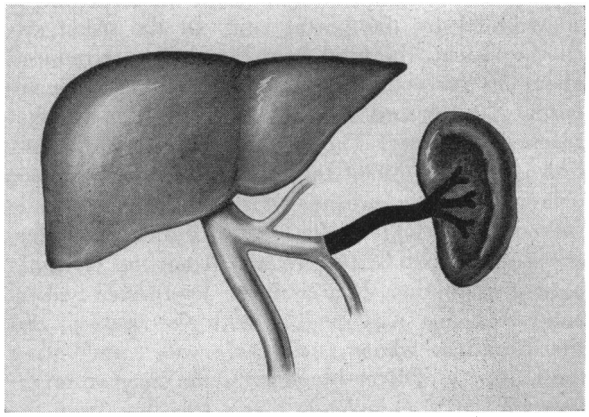

Figure 8.—Extrahepatic portal hypertension: Type No. 2. The splenic vein alone is thrombosed. The portal and superior mesenteric veins are patent.

mesenteric vein here, are unlikely to succeed since only the intestinal veins drained by this vessel will be decompressed. No relief will be afforded the venous return from the spleen, stomach or left colon.

Figure 5 is a splenoportagram taken in the case of a 34-year-old man with gastrointestinal bleeding and splenomegaly. The pronounced narrowing in the portal vein was due to chronic pancreatitis localized to the head of the gland. Splenorenal anastomosis successfully relieved the gastrointestinal bleeding.

Figure 6 is a splenoportagram in the case of a

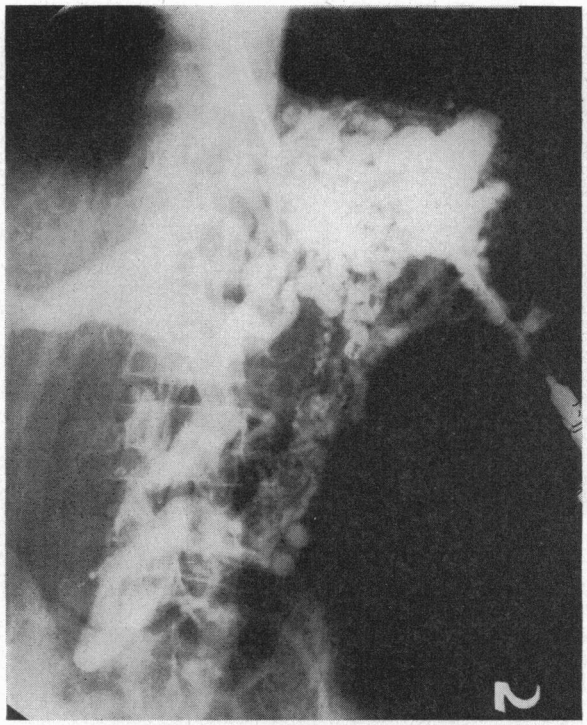

Figure 9.—Splenoportagram of 56-year-old man. A huge collateral circumvents the splenic vein obstruction to outline normally patent superior mesenteric and portal veins.

nine-month-old infant, with thrombosis of both the portal and superior mesenteric veins which was secondary to omphalitis following birth. Splenorenal shunt was successfully carried out. When the initial splenoportagram shows obstruction in the splenic vein, as it did in this infant, it is essential to perform a secondary portagram (Figure 7) via a jejunal mesenteric vein. This procedure is required to ascertain the status of the superior mesenteric and portal veins. If the splenic vein alone is obstructed, a lesser operative procedure is indicated. This will be clarified in succeeding paragraphs.

Type 2 (Figure 8): The splenic vein alone is thrombosed. In cases of this type the venous return from the majority of the abdominal viscera is flowing unobstructed in a normal fashion through the superior mesenteric and portal veins. The venous return from the spleen and perhaps part of the stomach is the only segment obstructed. In this situation, splenectomy alone, will suffice. The contribution of splenic blood to the varices, here, is probably in the neighborhood of 80 per cent or more. Figure 9 is a splenoportagram in the case of a 56-year-old man who had severe bleeding from varices. It was noted that a greatly enlarged collateral vessel circumvented the splenic vein obstruction to outline normally patent superior mesenteric and portal veins. This extensive collateral, however, had included the azygos and hemi-azygos systems

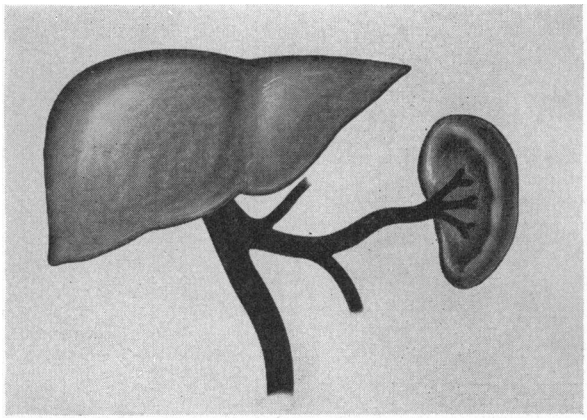

Figure 10.—Extrahepatic portal hypertension: Type No. 3. The major veins of the entire portal system are thrombosed.

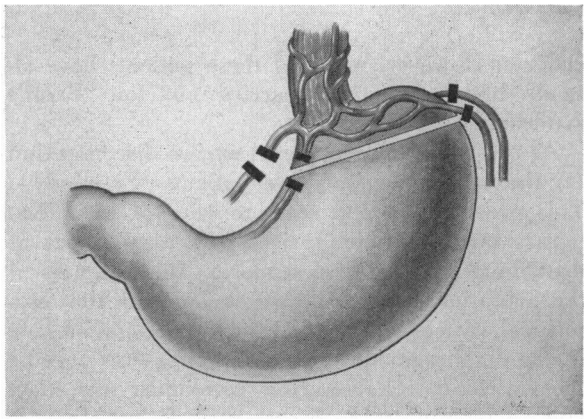

Figure 11.—Diagrammatic presentation of portal-azygos disconnection by division and immediate resuture of the gastric cardia.

in the mediastinum, resulting in esophagogastric varices. The etiologic process was a pseudocyst of the tail of the pancreas. No further bleeding occurred after splenectomy and distal pancreatectomy was carried out.

Type 3 (Figure 10): The major veins of the portal system are all thrombosed. No type of veno-venous shunt is possible in this situation. Shunts have been attempted between rather large collateral veins and the caval system, but they have failed since only a small segment of the splanchnic venous return was thus decompressed. In extrahepatic portal hypertension of this type it is necessary to operate directly on the varices. Three methods, each with minor variations, are available to accomplish this. The efficacy of each has yet to be determined. The three methods are segmental esophagogastrectomy, transesophageal ligation of the varices and portal azygos disconnection by transsection and resuture of the gastric cardia. Splenectomy may or may not be done as an adjunct to any of these pro-

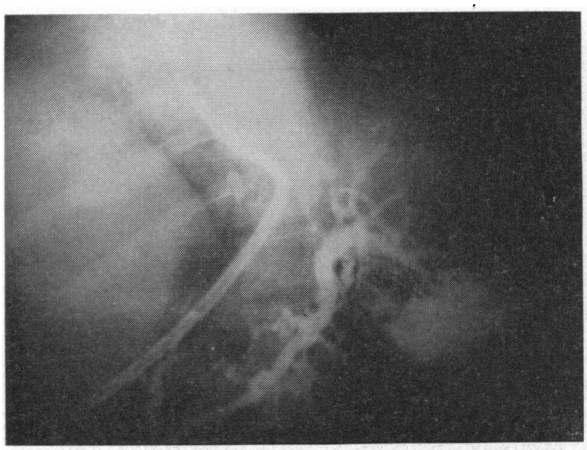

Figure 12.—Splenoportagram of 62-year-old woman. None of the major tributaries of the portal circulation are visualized. The one significant vein seen is the gastro-epiploic.

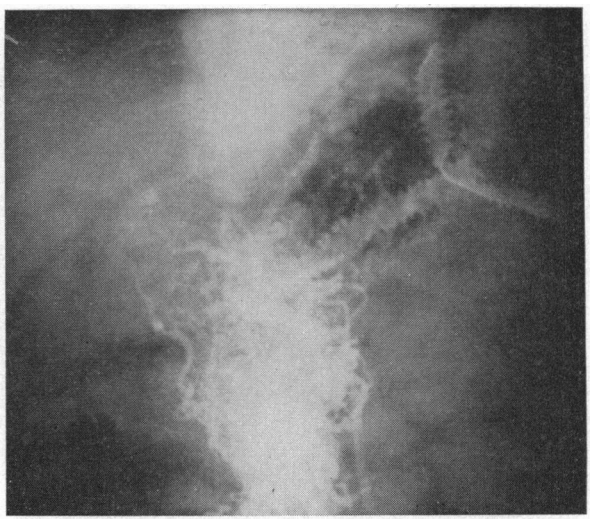

Figure 13.—Jejunal portagram of same patient shown in Figure 11. Neither the superior mesenteric nor the portal vein is visualized.

cedures. However, many of these patients have already had splenectomy carried out for "Banti's syndrome."

At present we prefer portal azygos disconnection for the management of these patients (Figure 11). This procedure would seem to offer at least temporary control of the varices with a minimum of disturbing postoperative sequelae. The rationale of the operation stems from the recognition that esophageal varices are filled from the abdominal side of the diaphragm. Interruption of this flow permits the varices to collapse. How permanent this effect will be remains to be ascertained. Pyloroplasty is required as an adjunct to this procedure in order to permit adequate gastric drainage, since vagotomy is technically unavoidable. Fortunately, vagotomy is desirable, since it interrupts the cephalic phase of gastric secretion and reduces the acid secretion of the stomach. Pyloroplasty accomplishes a similar result by reducing the antral phase of gastric secretion. It is believed by many observers that acid-peptic erosion of the esophageal mucosa is the initiating factor in varix bleeding. Our experience with this procedure is limited to two recent satisfactory results. In a recent personal communication, Mr. N. C. Tanner of St. James Hospital, London, stated that he is observing eight patients who have had this operation. None has bled and none has significant gastrointestinal complaints. The longest period of follow-up in this series is four years. It is readily apparent that further appraisal of this procedure is required.

Our preference for portal azygos disconnection has developed from dissatisfaction with the first two procedures listed. In three of four patients we operated upon, segmental esophagogastrectomy was followed by disabling esophagitis. Perhaps a more extensive proximal gastrectomy would offer greater

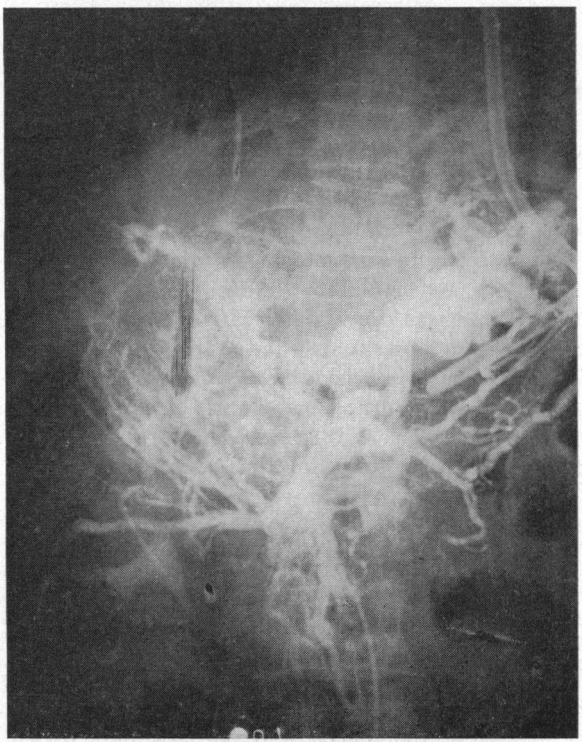

Figure 14.—Jejunal portagram of patient who had post-splenectomy bleeding. Collateral by-passing obliteration of portal, splenic and superior mesenteric veins is demonstrated.

protection in this regard. This operation, however, is a major one and a significantly higher operative mortality must be anticipated.

We have performed transesophageal ligation of varices in five patients. Three bled significantly after operation and two are now dead. The remaining two did not have further bleeding and were doing

satisfactorily when last observed, one of them 19 months and the other 27 months after operation. One of them had vagotomy, pyloroplasty and splenectomy in addition to ligation of the varices.

Figure 12 is a splenoportagram of this patient. The rather large vein leading from the spleen is not the splenic vein but rather the gastroepiploic vein. That the entire major portal system is obstructed is apparent in Figure 13, a portagram made with a jejunal mesenteric vein utilized for the injection. Pronounced collateral development without patency of the superior mesenteric or portal veins is demonstrated.

A similar picture of complete occlusion of all of the major radicals of the portal system is seen in Figure 14. The patient had had splenectomy four years previously for "Banti's syndrome." This portagram was obtained via a jejunal mesenteric vein.

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